

Citation:

Djoussé L, Pankow JS, Hunt SC, Heiss G, Province MA, Kabagambe EK, Ellison RC. Influence of saturated fat and linolenic acid on the association between intake of dairy products and blood pressure. *Hypertension*. 2006;48(2):335-41.

PubMed ID: [16801477](#)

Study Design:

Cross-sectional Study

Class:

D - [Click here](#) for explanation of classification scheme.

Research Design and Implementation Rating:

POSITIVE: See Research Design and Implementation Criteria Checklist below.

Research Purpose:

To examine the relation between dairy consumption and prevalent hypertension (HTN) among 4797 participants of the National Heart, Lung, and Blood Institute Family Heart Study, and to estimate prevalence odds ratios of HTN across categories of dairy consumption.

Inclusion Criteria:

- Participants in the NHLBI Family Heart Study
- Subjects with complete data on diet and blood pressure.

Exclusion Criteria:

- Excluded individuals without food frequency data.
- Excluded subjects because of probable errors on food frequency questionnaires (answers on the food frequency questionnaire judged by the interviewer as unreliable, or 18 items left blank on the dietary questionnaires or energy intake outside a priori ranges)

Description of Study Protocol:**Recruitment**

- The NHLBI Family Heart Study is a multicenter, population-based study designed to identify and evaluate genetic and nongenetic determinants of coronary heart disease (CHD), preclinical atherosclerosis, and cardiovascular risk factors.
- Families had been chosen randomly (a random group) or based on a higher-than-expected risk of CHD (a high-risk group) from previously established population-based cohort studies in Framingham, Mass; Forsyth County, NC; northwest suburbs of Minneapolis, Minn; and Salt Lake City, Utah. A family risk score relating the family's age- and sex-specific incidence of CHD to that expected in the general population the high-risk group.

Design: Cross-sectional study.

- In this study, dairy consumption was assessed through a staff-administered semiquantitative food frequency

questionnaire.

- The intake of specific nutrients (eg, calcium, magnesium, sodium, potassium, and caffeine) was computed by multiplying the frequency of consumption of an item by the nutrient content of specified portions.
- During a clinic visit at one of the field centers, a detailed medical and lifestyle history was obtained through interview, and laboratory measurements were done
- Subjects with complete data on diet and blood pressure are included in the analysis.
- Information on cigarette smoking, alcohol intake, education, and level of physical activity during the previous year was obtained by interview during the clinic visit. Diabetes mellitus was present if a subject was taking hypoglycemic agents, if a physician had told him/her that he/she has diabetes mellitus, or if fasting glucose levels were 7.0 mmol/L. Prevalent CHD was assessed by self-reported history of myocardial infarction, percutaneous transluminal coronary angioplasty, or coronary artery bypass graft.

Blinding used (if applicable): not applicable

Intervention (if applicable): not applicable

Statistical Analysis

- Gender-specific analyses: authors used generalized estimating equations to compute adjusted odds ratios (ORs) for prevalent HTN and adjusted mean blood pressure across quartiles of dairy consumption.
- The multivariable model controlled for age (deciles), gender, energy intake (deciles), body mass index, field center, total linolenic acid, saturated and monounsaturated fat, sodium, potassium, magnesium, caffeine, fiber, fruit and vegetable intake, education (3 groups), current alcohol intake (yes/no), current smoking (yes/no), and history of CHD and diabetes mellitus (yes/no).
- Additional adjustment for CHD risk group, physical activity, long-chain omega-3 fatty acids, polyunsaturated fatty acids, and dietary calcium had little effects on the point estimates (data not shown).
- Authors evaluated interactions by including the main effects and product terms in the regression model and compared model with and model without the interaction terms using partial likelihood ratio tests.
- For 3-way interaction, authors included 2-way product terms and main effects in the regression model.
- All of the analyses were performed using PC SAS (version 9.1).

Data Collection Summary:

Timing of Measurements

One time measurement.

Dependent Variables

- Blood pressure measured with random zero sphygmomanometer
- Prevalent hypertension

Independent Variables

- Dietary intake of dairy products assessed through staff-administered semiquantitative food frequency questionnaire

Control Variables

- Age
- Sex
- Energy intake
- Field center
- BMI
- Dietary linolenic acid
- Saturated and monounsaturated fat
- Sodium intake
- Potassium
- Caffeine
- Fiber, fruits and vegetables
- Cigarette smoking
- Alcohol intake
- Education

- Level of physical activity
- Percent energy from saturated fat

Description of Actual Data Sample:

Initial N: 4971

Attrition (final N): 4797; 45% were men

Age: Mean age : 52.2±13.7 years (range, 25 to 94 years)

Ethnicity: 4% were black

Other relevant demographics:

Anthropometrics

Location: Framingham, Mass; Forsyth County, NC; northwest suburbs of Minneapolis, Minn; and Salt Lake City, Utah

Summary of Results:

Key Findings

- There was an inverse association between dairy intake and prevalent HTN: odds ratios (95% CIs) were 1.0 (reference), 0.82 (0.64 - 1.05), 0.68 (0.53 - 0.89), and 0.62 (0.45 - 0.84), respectively, *P* for trend = 0.002.
- The association was independent of calcium intake and was mainly observed among subjects consuming fewer calories from saturated fat (*P* for interaction=0.014).
- Dairy consumption was inversely associated with systolic (*P* for trend=0.003) but not diastolic (*P* for trend=0.09) blood pressure.
- Although subjects consuming ≥ 2 servings per day of dairy products and higher total linolenic acid had the lowest prevalence odds of HTN, there was no evidence for interaction between linolenic acid and dairy consumption on HTN (*P* for interaction=0.65).
- Higher consumption of dairy products was associated with higher educational attainment; higher intake of fruits and vegetables, total linolenic acid, energy from saturated fat, magnesium, sodium, and potassium; lower consumption of caffeine; and lower percentage of current drinkers and smokers.

Other Findings

- Additional adjustment for dietary calcium made the inverse association between dairy and HTN slightly stronger: ORs of 1.0, 0.82 (95% CI, 0.63 to 1.06), 0.69 (95% CI, 0.52 to 0.92), and 0.61 (95% CI, 0.40 to 0.93) from the lowest to the highest quartile of dairy consumption, respectively (*P* for trend=0.02).
- A stronger inverse association between dairy and HTN among subjects consuming $<11.2\%$ saturated fat. Multivariable ORs were 1.0 (reference), 0.76, 0.53, and 0.46 from the lowest to the highest quartile of dairy intake, respectively (*P* for trend=0.001).
- The *P* value for interaction was statistically significant between saturated fat and dairy intake on HTN (*P*=0.014).
- The inverse association between dairy products and HTN was observed across all of the field centers (*P* for interaction between center and dairy product=0.4).

- There was an inverse association between calcium intake and prevalent HTN.
- There was a graded inverse association between dairy intake and SBP (P for trend=0.003) and little effects with DBP with a 2.6-mm Hg lower SBP comparing the highest with the lowest quartile of dairy intake in a multivariable model.
- Restricted to subjects whose energy intake from saturated fat was below the population median (11.2%) made the dairy- SBP association even stronger (3.5-mm Hg lower SBP in the highest dairy category compared with the lowest group; P for linear trend=0.01).
- Compared with subjects who consumed <2 servings per day of dairy products and <0.68 g per day of total linolenic acid (median), higher consumption of linolenic acid was associated with a 19% lower prevalence odds of HTN in a multivariable model; consumption of ≥ 2 servings per day of dairy products was associated with a 26% lower HTN prevalence odds; and higher intake of both dairy products and total linolenic acid was associated with a 35% lower prevalence odds of HTN.
- There was no evidence for a significant interaction between total linolenic acid and dairy consumption on HTN (P for interaction=0.65).
- There was little and nonsignificant association among age-, gender-, and energy-adjusted quartiles of combined eicosapentaenoic and docosahexaenoic acids and prevalent HTN.
- No evidence for effect modification of the dairy-HTN association by long-chain omega-3 fatty acids (P for interaction=0.28).

Prevalence ORs (95% CIs) of HTN According to Dairy and Saturated Fat (SFA) Consumption in the NHLBI Family Heart Study

Gender-, Age-, and Energy-Adjusted Quartiles of Dairy Consumption (Median Daily Servings)	% Energy from SFA <11.2		% Energy from SFA >11.2	
	Cases/N	Odds ORs (95% CI)*	Cases/N	ORs (95% CI)*
Q1 (0.4) low	131/677	1.0	65/446	1.0
Q2 (1.1)	109/626	0.76 (0.55 to 1.05)	84/601	0.94 (0.63 to 1.41)
Q3 (1.8)	84/628	0.53 (0.37 to 0.78)	94/641	1.03 (0.68 to 1.57)
Q4 (3.1) high	63/460	0.46 (0.29 to 0.74)	94/718	0.94 (0.55 to 1.60)
P for linear trend		0.001		0.89
P for interaction†			0.014	

HTN was defined as stages 1 and 2 of JNC VII or current treatment for high blood pressure. Median energy from saturated fat in the population was 11.2%.

*Adjusted for age, gender, field center, body mass index, energy intake, linolenic acid, saturated and monounsaturated fat, sodium intake, potassium, magnesium, caffeine, fiber, fruit and vegetable, education (3 groups), current drinking (yes/no), current smoking

(yes/no), and history of CHD and diabetes mellitus using generalized estimating equations.

† P value for interaction obtained using dairy consumption as a continuous variable

Author Conclusion:

In conclusion, our data indicate an inverse association between dairy consumption and prevalent HTN that was independent of dietary calcium, mainly among individuals consuming less saturated fat. This suggests that consumption of low-fat dairy products might be more beneficial for preventing HTN.

Reviewer Comments:

Large sample size and multicenter design.

Research Design and Implementation Criteria Checklist: Primary Research

Relevance Questions		
1.	Would implementing the studied intervention or procedure (if found successful) result in improved outcomes for the patients/clients/population group? (Not Applicable for some epidemiological studies)	Yes
2.	Did the authors study an outcome (dependent variable) or topic that the patients/clients/population group would care about?	Yes
3.	Is the focus of the intervention or procedure (independent variable) or topic of study a common issue of concern to nutrition or dietetics practice?	Yes
4.	Is the intervention or procedure feasible? (NA for some epidemiological studies)	Yes
Validity Questions		
1.	Was the research question clearly stated?	Yes
1.1.	Was (were) the specific intervention(s) or procedure(s) [independent variable(s)] identified?	Yes
1.2.	Was (were) the outcome(s) [dependent variable(s)] clearly indicated?	Yes
1.3.	Were the target population and setting specified?	Yes
2.	Was the selection of study subjects/patients free from bias?	Yes
2.1.	Were inclusion/exclusion criteria specified (e.g., risk, point in disease progression, diagnostic or prognosis criteria), and with sufficient detail and without omitting criteria critical to the study?	Yes
2.2.	Were criteria applied equally to all study groups?	Yes
2.3.	Were health, demographics, and other characteristics of subjects described?	Yes
2.4.	Were the subjects/patients a representative sample of the relevant population?	Yes
3.	Were study groups comparable?	Yes
3.1.	Was the method of assigning subjects/patients to groups described and unbiased? (Method of randomization identified if RCT)	Yes
3.2.	Were distribution of disease status, prognostic factors, and other factors (e.g., demographics) similar across study groups at baseline?	Yes
3.3.	Were concurrent controls used? (Concurrent preferred over historical controls.)	Yes

3.4.	If cohort study or cross-sectional study, were groups comparable on important confounding factors and/or were preexisting differences accounted for by using appropriate adjustments in statistical analysis?	Yes
3.5.	If case control or cross-sectional study, were potential confounding factors comparable for cases and controls? (If case series or trial with subjects serving as own control, this criterion is not applicable. Criterion may not be applicable in some cross-sectional studies.)	Yes
3.6.	If diagnostic test, was there an independent blind comparison with an appropriate reference standard (e.g., "gold standard")?	N/A
4.	Was method of handling withdrawals described?	Yes
4.1.	Were follow-up methods described and the same for all groups?	Yes
4.2.	Was the number, characteristics of withdrawals (i.e., dropouts, lost to follow up, attrition rate) and/or response rate (cross-sectional studies) described for each group? (Follow up goal for a strong study is 80%.)	Yes
4.3.	Were all enrolled subjects/patients (in the original sample) accounted for?	Yes
4.4.	Were reasons for withdrawals similar across groups?	N/A
4.5.	If diagnostic test, was decision to perform reference test not dependent on results of test under study?	N/A
5.	Was blinding used to prevent introduction of bias?	Yes
5.1.	In intervention study, were subjects, clinicians/practitioners, and investigators blinded to treatment group, as appropriate?	N/A
5.2.	Were data collectors blinded for outcomes assessment? (If outcome is measured using an objective test, such as a lab value, this criterion is assumed to be met.)	Yes
5.3.	In cohort study or cross-sectional study, were measurements of outcomes and risk factors blinded?	Yes
5.4.	In case control study, was case definition explicit and case ascertainment not influenced by exposure status?	N/A
5.5.	In diagnostic study, were test results blinded to patient history and other test results?	N/A
6.	Were intervention/therapeutic regimens/exposure factor or procedure and any comparison(s) described in detail? Were intervening factors described?	Yes
6.1.	In RCT or other intervention trial, were protocols described for all regimens studied?	N/A
6.2.	In observational study, were interventions, study settings, and clinicians/provider described?	Yes

6.3.	Was the intensity and duration of the intervention or exposure factor sufficient to produce a meaningful effect?	N/A
6.4.	Was the amount of exposure and, if relevant, subject/patient compliance measured?	N/A
6.5.	Were co-interventions (e.g., ancillary treatments, other therapies) described?	N/A
6.6.	Were extra or unplanned treatments described?	N/A
6.7.	Was the information for 6.4, 6.5, and 6.6 assessed the same way for all groups?	N/A
6.8.	In diagnostic study, were details of test administration and replication sufficient?	N/A
7.	Were outcomes clearly defined and the measurements valid and reliable?	Yes
7.1.	Were primary and secondary endpoints described and relevant to the question?	Yes
7.2.	Were nutrition measures appropriate to question and outcomes of concern?	Yes
7.3.	Was the period of follow-up long enough for important outcome(s) to occur?	N/A
7.4.	Were the observations and measurements based on standard, valid, and reliable data collection instruments/tests/procedures?	Yes
7.5.	Was the measurement of effect at an appropriate level of precision?	Yes
7.6.	Were other factors accounted for (measured) that could affect outcomes?	Yes
7.7.	Were the measurements conducted consistently across groups?	Yes
8.	Was the statistical analysis appropriate for the study design and type of outcome indicators?	Yes
8.1.	Were statistical analyses adequately described and the results reported appropriately?	Yes
8.2.	Were correct statistical tests used and assumptions of test not violated?	Yes
8.3.	Were statistics reported with levels of significance and/or confidence intervals?	Yes
8.4.	Was "intent to treat" analysis of outcomes done (and as appropriate, was there an analysis of outcomes for those maximally exposed or a dose-response analysis)?	N/A
8.5.	Were adequate adjustments made for effects of confounding factors that might have affected the outcomes (e.g., multivariate analyses)?	Yes
8.6.	Was clinical significance as well as statistical significance reported?	Yes

8.7.	If negative findings, was a power calculation reported to address type 2 error?	N/A
9.	Are conclusions supported by results with biases and limitations taken into consideration?	Yes
9.1.	Is there a discussion of findings?	Yes
9.2.	Are biases and study limitations identified and discussed?	Yes
10.	Is bias due to study's funding or sponsorship unlikely?	Yes
10.1.	Were sources of funding and investigators' affiliations described?	Yes
10.2.	Was the study free from apparent conflict of interest?	Yes

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